1990

Disease resistance in a wild system:hypoxylon canker of aspen

Michael E. Ostry

N.A. Anderson

Recommended Citation

Ostry, Michael E. and Anderson, N. A. Anderson. 1990. Disease resistance in a wild system: hypoxylon canker of aspen. Aspen Symposium '89 : Proceedings. General Technical Report NC-140. North Central Forest Experiment Station Forest Service, U.S. Department of Agriculture St. Paul, Minnesota. 237-241.

DISEASE RESISTANCE IN A WILD SYSTEM:HYPOXYLON CANKER OF ASPEN

Michael E. Ostry and Neil A. Anderson[1]

ABSTRACT.--The Hypoxylon canker-aspen disease system is an extremely complex host-parasite interaction that involves many biological and environmental factors. Our research of the infection process has helped to explain key variables in host resistance to this disease. Several types of resistance are important including resistance to infection, resistance to canker development, and spatial resistance that is influenced by stand density factors.

INTRODUCTION

Plant pathologists have the responsibility of screening the world's food and fiber crops to identify genes that confer disease resistance. The use and misuse of these genes have far-reaching consequences. Most of the host-pathogen systems in agriculture involve either an introduced host or a pathogen, or both. Much of what we know about disease resistance is from situations where one or both partners in the parasitic relationship have been introduced.

Host-parasite interactions of indigenous systems may differ from systems in which one or both partners have been introduced. For example, aeciospores of the introduced white pine blister rust fungus (Cronartium ribicola) can spread for miles from white pine to ribes plants (Anderson 1973), but aeciospores of the indigenous sweetfern rust (C. comptoniae) travel only a few feet from the native jack pine to the native sweetfern or sweet gale (Anderson and French 1964). Urediospores of the introduced wheat stem rust fungus (Puccinia graminis var. tritici) can spread in great leaps from Mexico to Canada (Stakman and Harrar 1957), while some rusts on native plants in northern latitudes complete their life cycle on an alternate host only a few meters away (Savile 1976).

The best genetic explanation of host-pathogen interactions is the gene-for-gene theory of Flor (1956). In this hypothesis, genes for virulence in the pathogen are matched against specific disease resistance genes in the host. However, all host-pathogen systems that have conformed to this theory have included a highly cultivated host plant (Barrett 1985). Most biologists now consider Flor's theory a genetic critique of modern agriculture where vast acreages are planted with a crop that has limited genetic diversity. In modern agriculture, breeders must meet the challenge of the ever-changing pathogen populations that overcome host resistance by deploying new resistance genes.

To gain information on the nature of disease resistance in a wild system we chose to study the aspen-Hypoxylon canker disease system. Populus tremuloides Michx. has the widest range of any tree species in our hemisphere and is rich in genetic diversity. Aspen clones differ in many traits (Cheliak and Pitel 1984, Hyun et al. 1987), including their resistance to diseases (Capony and Barnes 1974). Clones of 200 acres or more occur in western U.S., and clones of 0.1 to 3 acres occur in the Lake States (Kemperman and Barnes 1976).

[1]Michael E. Ostry, North Central Forest Experiment Station and Neil A. Anderson, University of Minnesota, St. Paul, 55108. Scientific Journal Series Paper 17376, Minnesota Agricultural Experiment Station, St. Paul

Hypoxylon mammatum (Wahl.) Mill. is the most destructive pathogen of young aspen in the Lake States. Much has been learned about the aspen-Hypoxylon relationship, but many critical gaps must be filled before effective management strategies can be developed to minimize the impact of this disease (Manion and Griffin 1986). One missing piece of information is how trees become infected.

The objective of our research was to study the infection process of Hypoxylon in aspen. Our goals were to determine the nature of resistance of some aspen clones to the fungus and to reconcile laboratory research results on the host and pathogen with field biology studies.

DISEASE CYCLE

Many investigators have studied the infection process of Hypoxylon. Classical signs and symptoms of the canker disease include a dead branch or dead branch stub in the center of the canker and marbleized yellow-black decayed xylem tissue behind the canker face. Although the fungus is a wound parasite (Bier 1940), some infections can be found on trees with no observable injury.
Studies have shown the green layer in the aspen bark contains pyrochatechol, two glycocides, and a phenol that are fungistatic to the spores and mycelium of Hypoxylon (French and Oshima 1959, Hubbes 1964, 1966). The fungus produces a toxin that prevents callus formation and detoxifies the fungistatic chemicals in the green bark layer (Schipper 1978). H. mammatum destroys phloem tissues, but has limited enzymes to break down sucrose, the sugar transported in the phloem (Anderson and Schipper 1975). The fungus does, however, have enzymes that break down cellulose and cause wood decay.

In our studies, we have associated insect oviposition wounds with infection of aspen by Hypoxylon. Wounds made by cicada (Magicicada septendecim L.), poplar-gall saperda, (Saperda inornata Say), and tree hoppers (Telamona tremulata Ball) are entry courts for Hypoxylon (Anderson et al. 1979, Ostry and Anderson 1983, 1986). We have examined cross-sections of naturally infected aspen S. inornata galls that had only slight external disease symptoms. In all cases the xylem tissue showed advance symptoms of decay and only small portions of the phloem were decayed. Scanning electron microscopy revealed that the fungus formed extensive aggregated sheets of hyphae before invading the phloem. Cankers have developed on aspen branches inoculated with Hypoxylon ascospores through saperda galls (Anderson and Ostry 1983), indicating that ascospore infection of insect wounds may be a major means of entry.

The relationship between certain wood-boring insects of aspen and infection by Hypoxylon has provided us with an insight into the infection process, the stand density-canker incidence phenomenon, the pattern of infection on individual trees, fluctuation in canker incidence, the distribution of the disease in the Lake States, and host resistance to this disease. Downy woodpeckers (Picoides pubescens) frequently forage on and around cankers on aspen in search of larvae of various wood-boring insects. Woodpeckers also forage on Saperda galls, resulting in additional wounds on aspen branches as the birds extract larvae, and perhaps, serve as vectors for the fungus (Ostry et al. 1982).

PATHOGEN VARIABILITY

The growth characteristics and virulence of several H. mammatum isolates collected throughout the north central U.S. varied greatly, indicating the presence of several biotypes (Anderson and Schipper 1975). All of the isolates were pathogenic, but, large differences were present in growth rate of the fungus in vitro and in canker elongation when they were inoculated into aspen. In our work, up to one-third of the isolates obtained from single ascospore cultures were slow-growing mutant types. However, when we isolated from cankers in the field, we obtained only wild-type fast-growing isolates. This indicates that the fungus has an effective way of eliminating mutant types and only fast-growing types survive in nature.

HOST RESISTANCE

To study the apparent genetic differences in aspen susceptibility to Hypoxylon, we established two aspen plantations in Minnesota and Wisconsin. Controlled crosses were made of parent trees selected from highly infected clones and canker-free clones throughout Minnesota. Trees from 85 different crosses were planted. Parent trees propagated from root cuttings were also included in the plantings. Nearly 2,000 trees have been planted since 1965. The incidence of Hypoxylon canker varies between progeny of the various parents. We identified about 50 trees that are superior in form and insect and disease resistance. These trees are being clonally propagated by using tissue culture techniques, and they will be field tested on several different sites.

Resistance of aspen to Hypoxylon includes resistance to infection and subsequent canker development. Superimposed on these resistance mechanisms is the inherent resistance provided to aspen growing in dense stands. This type of resistance has been called spatial resistance, which includes the many environmental and biotic factors associated with stand density that affect disease expression toward decreased disease incidence (McNabb et al. 1982).

STAND DENSITY

Several investigators have associated higher canker incidence with low stand density and trees along stand edges (Day and Strong 1959, Anderson 1964, Anderson and Anderson 1968, Bruck and Manion 1980, Anderson and Martin 1981). In a study designed to determine the relation between the incidence of Hypoxylon canker and the presence of branches on aspen, pruned trees had fewer lower bole cankers (Ostry and Anderson 1979). Aspen in thinnned or understocked stands have branches that persist longer than in dense stands, increasing the chance of being infected by Hypoxylon. Other factors associated with stand density that have been suggested as favoring infection include decreased soil moisture and nutrients, and increased sunlight and air movement in open stands. The task of determining relations between site variables is further complicated by clonal differences in susceptibility to the fungus. We have data that indicate that the clones in study plots result in greater differences in canker incidence than the thinning and fertilization treatments applied.[2]

SUMMARY AND MANAGEMENT IMPLICATIONS

Our studies over the past two decades have confirmed that resistance in aspen to Hypoxylon is complex. A specific type of wound is required for infection by the fungus, and certain environmental conditions are required for disease development. The tree responds to wounds by forming callus that closes the wounds, preventing infection or inhibiting canker expansion. Clonal differences in these responses are common and indicate that it may be possible to select superior genotypes. Stand density influences the incidence of the disease through several interacting factors, and this spatial resistance may provide a possible management strategy to minimize the disease impact.

Because aspens grow as a mosaic of relatively small clones in the Lake States, clone identification is important. This is especially critical in research and inventory plots to account for variability in treatment effects, data on tree growth, or insect and disease impact. Several tree traits can be used to distinguish clones (Barnes 1969). We are also using aerial photography to assist in the delineation of clone boundaries within our research plots. Silvicultural methods for expanding clones of superior aspen may be important in the future as part of intensive management strategies for the aspen resource.

---

[2]M.E. Ostry and N.A. Anderson, North Central Forest Experiment Station, St. Paul, MN., unpublished data.

Tissue culture techniques are being developed that will allow clonal multiplication of selected aspen genotypes for planting. By planting the same aspen clone on several different sites, we will learn how site factors influence aspen growth and the incidence and severity of insects and diseases. At the present time, clonal variability makes it difficult to sort out the important factors that contribute to the resistance or susceptibility of aspen to damaging agents.

Much research remains to be done before we can develop sound intensive management practices for aspen. With the increasing interest in aspen come the challenge and opportunity to increase the quantity and quality of our stands. With a multidisciplinary team approach, including people skilled in silviculture, genetics, insect and disease control, and use of new tissue culture and molecular techniques, we can begin to improve upon our management of aspen and maintain this important resource for its many uses.

LITERATURE CITED

Anderson, R.L. 1964. Hypoxylon canker impact on aspen. Phytopathology 54:253-257.

Anderson, R.L. 1973. A summary of white pine blister rust research in the Lake States. North Central Forest Experiment Station, St. Paul, MN. USDA For. Serv. Gen. Tech. Rep. NC-6. 12 p.

Anderson, G.W., and R.L. Anderson. 1968. Relationship between density of quaking aspen and incidence of Hypoxylon canker. Forest Sci. 14:107-112.

Anderson, N.A., and D.W. French. 1964. Sweetfern rust on jack pine. J. For. 62:467-471.

Anderson, G.W., and M.P. Martin. 1981. Factors related to incidence of Hypoxylon cankers in aspen and survival of cankered trees. Forest Sci. 27:461-476.

Anderson, N.A., and M.E. Ostry. 1983. Galleries of Saperda inornata as infection courts of Hypoxylon mammatum on trembling aspen. Phytopathology 73:836.

Anderson, N.A., M.E. Ostry, and G.W. Anderson. 1979. Insect wounds as infection sites for Hypoxylon mammatum on trembling aspen. Phytopathology 69:476-479.

Anderson, G.W., and A.L. Schipper, Jr. 1975. Variation among isolates of Hypoxylon mammatum. Eur. J. For. Path. 5:216-224.

Barnes, B.V. 1969. Natural variation and delineation of clones of Populus tremuloides and P. grandidentata in northern lower Michigan. Silvae Genet. 18:130-142.

Barrett, J. 1985. The gene-for-gene hypothesis: parable or paradigm. P. 215-225 *in* Ecology and Genetics of Host-Parasite Interactions. Rollinson, D., and R.M. Anderson, eds. Academic Press.

Bier, J.E. 1940. Studies in forest pathology lll. Hypoxylon canker of poplar. Can. Dept. Agric. Publ. 691. 40 p.

Bruck, R.I., and P.D. Manion. 1980. Interacting environmental factors associated with the incidence of Hypoxylon canker on trembling aspen. Can. J. For. Res. 10:17-24.

Capony, J.A., and B.V. Barnes. 1974. Clonal variation in the incidence of Hypoxylon canker on trembling aspen. Can. J. Bot. 52:1475-1481.

Cheliak, W.M., and J.A. Pitel. 1984. Electrophoretic identification of clones in trembling aspen. Can. J. For. Res. 14:740-743.

Day, M.W., and F.C. Strong. 1959. A study of hypoxylon canker on aspen. Mich. Agric. Exp. Stn. Q. Bull. 41:870-877.

Flor, H.H. 1956. The complementary genic systems in flax and flax rust. Advances in Genetics 8:29-54.

French, D.W., and N. Oshima. 1959. Host bark characteristics and infections by Hypoxylon pruinatum (Klot.) Cke. For. Sci. 5:255-258.

Hubbes, M. 1964. New facts on host-parasite relationships in the Hypoxylon canker of aspen. Can. J. Bot. 42:1489-1494.

Hubbes, M. 1966. Inhibition of Hypoxylon pruinatum (Klotzshe) Cke. by aspen bark meal and the nature of active extractives. Can. J. Bot. 44:365-386.

Hyun, J.O., O.P. Rajora, and L. Zsuffa. 1987. Genetic variation in trembling aspen in Ontario based on isozyme studies. Can. J. For. Res. 17:1134-1138.

Kemperman, J., and B.V. Barnes. 1976. Clone size in American aspens. Can. J. Bot. 54:2603-2607.

Manion, P.D., and D.H. Griffin. 1986. Sixty-five years of research on Hypoxylon canker of aspen. Plant Disease 70:803-808.

McNabb, H.S. Jr., R.B. Hall, and M.E. Ostry. 1982. Biological and physical modifications of the environment in short rotation tree crops and the resulting effect upon the host-parasite interactions in short-rotation tree crops. P. 60-71 *in* Proceedings, Third International Workshop on the Genetics of Host-Parasite Interactions in Forestry: Resistance to Diseases and Pests in Forest Trees. H.M. Heybroek, B.R. Stephan, and K. von Weissenberg, eds. Pudoc, Wangeningen, the Netherlands.

Ostry, M.E., and G.W. Anderson. 1979. Hypoxylon canker incidence on pruned and unpruned aspen. Can. J. For. Res. 9:290-291.

Ostry, M.E., and N.A. Anderson. 1983. Infection of trembling aspen by Hypoxylon mammatum through cicada oviposition wounds. Phytopathology 73:1092-1096.

Ostry, M.E., and N.A. Anderson. 1986. Infection of aspen by Hypoxylon mammatum through treehopper wounds. Phytopathology 76:957.

Ostry, M.E., K. Daniels, and N.A. Anderson. 1982. Downy woodpeckers--a missing link in a forest disease life cycle? The Loon 54:170-175.

Savile, D.B.O. 1976. Evaluation of the rust fungi (Uredinales) as reflected by their ecological problems. Evol. Biol. 9:137-207.

Schipper, A.L., Jr. 1978. A Hypoxylon mammatum pathotoxin responsible for canker formation in quaking aspen. Phytopathology 68:866-872.

Stakman, E.C., and J.G. Harrar. 1957. Principles of Plant Pathology. Ronald Press, New York. 581 p.